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Cardiac Manifestations of Cocaine Abuse: A Cross-Sectional Study of Asymptomatic Men With a History of Long-Term Abuse of "Crack" Cocaine

SIMON CHAKKO, MD, FACC, ALAND FERNANDEZ, MD, THOMAS A. MELLMAN, MD, FERNANDO J. MILANES, MD, KENNETH M. KESSLER, MD, FACC, ROBERT J. MYERBURG, MD, FACC

Miami, Florida

Objectives. The objective of this study was to evaluate the prevalence of cardiac abnormalities in young, asymptomatic long-term "crack" cocaine abusers.

Background. Although the cardiac complications of cocaine abuse have received widespread attention, the prevalence of cardiac abnormalities in asymptomatic long-term cocaine abusers is unknown.

Methods. History, physical examination, electrocardiogram (ECG) and echocardiogram were performed in 52 consecutive long-term cocaine abusers admitted to a drug rehabilitation program. Findings were compared with those in 14 age-matched normal volunteers and 14 age-matched normotensive patients admitted to a psychiatric service who had a pattern of smoking and alcohol consumption similar to that of the study patients.

Results. The ECG findings were abnormal in 29% of cocaine abusers, and included nonspecific ST-T wave changes in 15%, abnormal ST segment elevation in 10%, old inferior infarction in 2%, old anteroseptal infarction in 2% and abnormal precordial R wave progression in 10%. When compared with normal volunteers and control patients, cocaine abusers had increased left ventricular posterior wall thickness (1.12 vs. 0.76 and 0.85 cm, respectively, $p < 0.0001$), increased septal thickness (1.13 vs. 0.76 and 0.86 cm, $p < 0.001$) and higher left ventricular mass index (142 vs. 84 and 94 g/m^2, $p < 0.0001$). Left ventricular diastolic filling variables did not differ significantly among the three groups. Diastolic filling variables were similar in cocaine abusers with and without left ventricular hypertrophy, and the prevalence of left ventricular hypertrophy did not differ significantly between those who used no alcohol or <35 ml/week of alcohol and those who consumed ≥500 ml/week of alcohol. Left ventricular segmental wall motion abnormalities were present in 11 subjects (21%) and the ejection fraction was decreased (<0.45) in 2 (4%).

Conclusions. Electrocardiographic and echocardiographic abnormalities are common in long-term cocaine abusers. Despite the frequent occurrence of left ventricular hypertrophy, Doppler-derived diastolic filling pattern was not altered. Concomitant alcohol use did not affect the prevalence of these abnormalities.

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It is estimated that 30 million Americans have used cocaine, 5 million use it regularly and every day 5,000 use it for the first time (1,2). Cocaine became more pure, less expensive and easier to obtain during the 1980s, a period during which reports of cocaine-related cardiovascular events increased (3). Cocaine-related myocardial ischemia, infarction, coronary spasm, pulmonary edema, cardiac arrhythmias, sudden death, myocarditis and dilated cardiomyopathy have been reported (1,3,4). Cocaine blocks the presynaptic reuptake of the neurotransmitters norepinephrine and dopamine, producing an excess of transmitter at the postsynaptic receptor sites. Activation of the sympathetic nervous system by this mechanism produces vasoconstriction, an abrupt rise in arterial blood pressure, tachycardia and a predisposition to ventricular arrhythmias (1).

These reports of cardiac complications of cocaine have received widespread attention in scientific publications and the lay press. However, these findings are based on reports of isolated cases, on series of cases in which patients presented with a major cardiovascular complication and on autopsy studies (5,6). Although cocaine abuse has reached epidemic proportions, the number of complications seen clinically is very small; only 58 cases of myocardial infarction have been reported in English language publications (4). Thus, it is possible that cocaine causes clinically evident cardiac complications in only a small number of abusers, and cardiac abnormalities induced by cocaine may be the exception rather than the rule. However, the long-term cardiac complications of long-term cocaine abuse have not been studied prospectively and their prevalence is unknown. It is also possible that many cocaine abusers have cardiac abnormalities that are not recognized until a major cardiovascular event occurs. The purpose of this study was to evaluate

From the Departments of Medicine and Psychiatry, Miami Veterans Affairs Medical Center, and the University of Miami School of Medicine, Miami, Florida.

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Address for correspondence: Simon Chakko, MD, 111-A, Cardiology, Veterans Affairs Medical Center, Miami, Florida 33176.

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prospectively the prevalence of cardiac abnormalities in long-term cocaine abusers and to compare it with that in age-matched normal control subjects.

Methods

Study group. Fifty-five consecutive patients <45 years old with a history of long-term cocaine abuse were admitted to the drug dependence treatment program of the Miami Veterans Affairs Medical Center for immediate drug detoxification. Patients with hypertension, a history of any cardiovascular disease or significant medical conditions were excluded. Patients were initially interviewed by a psychiatrist, and the admission criteria were acute cocaine intoxication, history of long-term cocaine abuse and low probability of abstinence without hospitalization. The frequency and duration of cocaine, alcohol and other substance abuse were determined. The duration and amount of cocaine used (in dollars spent per day) and method of drug ingestion were recorded. Alcohol consumption was estimated from the number of cans of beer, glasses of wine or drinks of liquor that patients reported they drank each week. Total alcohol intake was estimated in ml/week as 16.7 × beer cans/week + 17.0 × wine glasses/week + 19.1 × liquor drinks/week (7). Blood pressure, hematocrit, blood cell count and serum electrolytes were recorded. Urine was analyzed for the presence of amphetamine, barbiturates, cannabis, cocaine, methadone and opiates.

Control groups. An age-matched control group consisting of 14 normal, healthy volunteers with no history of drug use, hypertension or any other illness was recruited from the hospital physicians and nurses. Another age-matched control group consisting of 14 patients admitted to the psychiatric service without a history of nonpsychiatric illness, hypertension or illicit drug use was also studied. Alcohol consumption was estimated by the method described for the cocaine abusers. All subjects in the study including the control groups were men and none had a history of weight lifting or endurance exercise training. The study was approved by the research and development committee of the Miami Veterans Affairs Medical Center, and informed consent was obtained from all participants.

Protocol. A patient history was recorded and physical examination and an electrocardiogram (ECG) were performed. M-mode, two-dimensional color flow and pulsed wave Doppler and Doppler color flow echocardiography were performed with a Hewlett-Packard (model 7020 A) imaging system. Accurate calibration was confirmed with a phantom. M-mode echocardiograms were recorded on strip chart recorder; measurements were made according to the recommendations of American Society of Echocardiography, and posterior wall and septal thickness were measured at the onset of the QRS complex (8). Left ventricular mass was calculated by the Penn cube method described by Devereux et al. (9). Left ventricular hypertrophy was defined as left ventricular mass index >134 g/m^2 body surface area. Two-dimensional echocardiograms were recorded on video tape and analyzed with an off-line computer system (Microsonics); measurements were made according to the recommendations of the American Society of Echocardiography (10). Ejection fraction was estimated by the single-plane area-length method from the apical four-chamber view or the fractional area change method form the parasternal short-axis view; the view with the best endocardial definition was used. Segmental wall motion abnormalities were evaluated visually; when the presence of wall motion abnormality was equivocal, the image was digitized and played back in a cine loop format. The right atrium was measured in its short and long axis and the right ventricle in its short axis from the apical four-chamber view obtained with two-dimensional echocardiography, using previously described methods (10,11).

Left ventricular diastolic filling was evaluated by pulsed Doppler study of mitral inflow. The sample volume was positioned just inferior to the level of the mitral anulus between the mitral leaflets. Signals were recorded on video tape and five cycles with clear signals were traced with the use of an off-line computer system (Microsonics). Measurements from the five cycles were averaged. The peak of the initial velocity, representing the early filling phase (E), and the late filling phase (A), representing atrial contraction, were measured (12). The acceleration half-time (time for the early peak velocity to reach its peak from half its peak value) and deceleration half-time (time for the early flow velocity to decrease from its peak to half the peak value) were measured by previously described techniques (13). Peak filling rate normalized to mitral stroke volume was derived with the formula of Bowman et al. (14). Echocardiograms were interpreted independently by two experienced observers, and only the abnormalities reported by both observers were included. To study the intraobserver and interobserver variations in M-mode echocardiographic and Doppler evaluations, measurements were made twice in 15 studies (21%) by the primary observer and by the second observer in a blinded fashion. No significant difference was found between the paired measurements.

Statistical analysis. Statistical analysis was performed with use of a commercially available software package (Graphpad-Instat). Results are reported as mean value ± 1 SD. Differences between the mean values of two sets of measurements were analyzed by paired or unpaired two-sample *t* test, and a two-tailed p value < 0.05 was considered significant. When the standard deviations of the groups were significantly different, the Mann-Whitney nonparametric test was used. When the three groups were compared with each other, one-way analysis of variance (ANOVA) was used. The p value (with Bonferroni correction) < 0.05 was considered significant (15).

Results

Patient characteristics. Of the 55 patients enrolled in the study, 2 had inadequate echocardiograms and 1 patient was

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Table 1. Electrocardiographic Findings in the 52 Patients Treated for Long-Term Cocaine Abuse

	No.	%
Rhythm		
Normal sinus rhythm	40	77
Sinus bradycardia	11	21
Ectopic atrial rhythm	1	2
Axis and intervals		
Intraventricular conduction delay	2	4
First-degree AV block	1	2
Left axis deviation	1	2
QRS complex		
Increased QRS voltage	12	23
Poor precordial R wave progression	5	10
Right bundle branch block	1	2
Old inferior infarction	1	2
Old anterior infarction	1	2
ST segment and T wave		
ST segment elevation (early repolarization)	26	50
Nonspecific ST-T abnormality	8	15
ST segment elevation (possible ischemia)	5	10

AV = atrioventricular.

found to have deep vein thrombosis; these 3 patients were excluded. None of the 14 normal volunteers or 14 control patients were excluded after enrollment. All 80 study participants were male, and the mean age in the three groups was similar (cocaine abusers, 36 ± 5 years; normal volunteers, 33 ± 4 years, and control patients, 34 ± 5 years; p = NS). Results of physical examination of the heart were normal in all 80 subjects. Among the 52 cocaine abusers, 41 (79%) were black, 9 (17%) white and 2 (4%) Hispanic. Of the 14 normal volunteers, 6 (43%) were Hispanic, 4 (28%) white, 3 (21%) Asian and 1 (7%) was black. Among the 14 control patients, 6 (43%) were white, 4 (28%) Hispanic and 4 (28%) black. No subject had a history of hypertension, and all had normal blood pressure measurements. Systolic blood pressure was 119 ± 12, 125 ± 5 and 120 ± 11 mm Hg (p = NS) in the cocaine abusers, normal volunteers and control patients, respectively. Diastolic pressure was 72 ± 9, 75 ± 6 and 73 ± 8 mm Hg respectively (p = NS). All cocaine abusers admitted to using "crack" cocaine. In addition, 9 (17%) snorted cocaine intranasally, 3 (6%) injected cocaine intravenously, 48 (92%) smoked tobacco, 41 (78%) drank varying amounts of alcohol and 1 (2%) injected heroin intravenously. Results of urinalysis for illicit drugs performed on admission were available in 44 (85%) of cocaine abusers; the data revealed metabolites of cocaine in 33 (75%), of cannabis in 8 (18%) and of opiates in 3 (7%). None of the normal volunteers smoked cigarettes and all consumed <35 ml of alcohol/week. Among the control patients, 87% smoked tobacco, 43% did not use alcohol or imbibed <35 ml/week and 57% drank 234 ml/week of alcohol.

Electrocardiographic findings in the 52 cocaine abusers (Table 1). ST segment elevation was present in 31 (60%) of the 52 patients; in 26 it was due to early repolarization and in 5 it did not have the typical features of early repolarization and was suggestive of ischemia. Old inferior wall infarction was noted in one subject (2%) and old anteroseptal infarction in another (2%). Poor precordial R wave progression consistent with old septal infarction was noted in five patients (10%). Complete right bundle branch block was present in 1 patient (2%), left axis deviation in 1 (2%), intraventricular conduction delay in 2 (4%), first-degree atrioventricular (AV) block in 1 (2%), sinus bradycardia in 11 (21%), ectopic atrial rhythm in 1 (2%) and nonspecific ST-T wave abnormalities in 8 (15%). Increased QRS voltage was present in 12 patients (23%). If sinus bradycardia, isolated increased QRS voltage and typical early repolarization were classified as normal variants, 15 (29%) of the cocaine abusers had abnormal ECG findings.

Echocardiographic features of all study subjects (Table 2). The most prevalent abnormality was left ventricular hypertrophy. The criterion for left ventricular hypertrophy in men, a left ventricular mass index >134 g/m^2 (16), was met in 28 (54%) of cocaine abusers; none of the normal volunteers or control patients met this criterion. In addition, values for mean left ventricular wall thickness, mass and mass index (corrected for body surface area) were all markedly higher in the cocaine abusers than in the control groups. The mean left ventricular mass index in the cocaine abusers was 142 ± 33 g/m^2. Only one of the control patients had hypertrophy of the septum and posterior wall (>1.1 cm). Among the 52 cocaine abusers, 41 were black and 11 were white or Hispanic; the left ventricular mass index in these subgroups was 140 ± 38 and 145 ± 13 g/m^2, respectively (p = NS). Left ventricular systolic function measured by ejection fraction was normal and similar in the three groups. Fractional shortening measured from the M-mode echocardiogram was also normal in all groups, but the value was significantly higher in the cocaine abusers than in the two control groups ($p < 0.05$). A dilated left ventricle with global hypokinesia was present in one cocaine abuser (1.9%). Segmental wall motion abnormalities were detected in 11 patients (21%) in the cocaine group, septal wall hypokinesia in 4, inferior wall hypokinesia in 4, posterior wall hypokinesia in 1, septal wall akinesia in 1 and inferior wall akinesia in 1. Two other wall motion abnormalities that were reported by only one observer were excluded from analysis. Left ventricular ejection fraction was decreased (<0.45) in two (4%).

Adequate pulsed wave Doppler echocardiograms were obtained in 48 patients. Left ventricular filling variables are shown in Table 3. Despite the presence of hypertrophy, the left ventricular diastolic filling pattern of cocaine abusers was not significantly different from that of men in the control groups. The diastolic filling variables did not differ significantly between cocaine abusers with and without left ventricular hypertrophy (Table 4). The echocardiographic abnormalities noted in the three groups are summarized in Table 5. Right atrial and right ventricular dimensions were larger in cocaine abusers than in the other groups (Table 2);

Table 2. Echocardiographic Measurements in Cocaine Abusers (Group 1), Normal Volunteers (Group 2) and Control Patients (Group 3)

	Group 1 (n = 52)	Group 2 (n = 14)	Group 3 (n = 14)	F	p Value
RA short axis (cm)	3.6 ± 0.45*‡	2.9 ± 0.63	3.1 ± 0.37	15.23	0.0001
RA long axis (cm)	4.2 ± 0.49‡	3.9 ± 0.40	3.8 ± 0.37	5.49	0.005
RV short axis (cm)	3.1 ± 0.65	2.8 ± 0.51	2.9 ± 0.50	1.64	NS
Left atrium (cm/m^2)	1.9 ± 0.53	1.8 ± 0.56	2.0 ± 0.12	0.58	NS
LV end-diastole (cm/m^2)	2.6 ± 0.49	2.7 ± 0.24	2.6 ± 0.26	0.20	NS
LV end-diastole (cm)	5.2 ± 0.58	5.2 ± 0.48	5.1 ± 0.57	0.18	NS
LV end-systole (cm)	2.7 ± 0.52	2.9 ± 0.49	2.9 ± 0.12	1.64	NS
Fractional shortening	0.48 ± 0.08§	0.42 ± 0.05	0.41 ± 0.07	7.06	0.001
LV ejection fraction	0.64 ± 0.12	0.63 ± 0.04	0.62 ± 0.06	0.17	NS
Septum (cm)	1.13 ± 0.17†	0.76 ± 0.01	0.86 ± 0.15	39.25	0.001
Posterior wall (cm)	1.12 ± 0.14†	0.76 ± 0.01	0.85 ± 0.14	51.79	0.0001
LV mass (g)	272 ± 67†	159 ± 84	183 ± 94‖	16.76	0.0001
LV mass index (g/m^2)	142 ± 33†	84 ± 21	94 ± 25‖	28.75	0.0001

The last column shows the significance of the differences between the three groups using analysis of variance. Significant Bonferroni p values with intergroup comparison are: *, †$p < 0.001$, Group 1 versus Group 2 and versus Groups 2 *and* 3, respectively. ‡, §$p < 0.05$, Group 1 versus Group 3 and versus Groups 2 *and* 3, respectively. ‖$p < 0.01$, Group 2 versus Group 3 (shown under Group 3). LV = left ventricular; RA = right atrial; RV = right ventricular.

however, only the difference in the right atrial dimension was statistically significant ($p < 0.001$).

Effect of concomitant cocaine and alcohol abuse. The cocaine abusers were arbitrarily classified into three groups according to the amount of alcohol consumed. Eighteen were classified as having heavy consumption of alcohol (500 ml/week), whereas 19 did not use alcohol or consumed <30 ml/week and 14 used an intermediate amount. Among these three groups of patients, there were no significant differences in left ventricular mass, mass index, ejection fraction or diastolic dimension (Table 6). The amount of cocaine used was estimated (in dollars spent per day), but the level of consumption was not consistent and often varied with the availability of money. Thus, the estimated amount of cocaine used was not a reliable measure and did not correlate with the echocardiographic measurements.

Echocardiographic findings and the electrocardiogram in cocaine abusers. Among the 52 cocaine abusers, 31 had at least one echocardiographic abnormality and 17 of the 31 had at least one ECG abnormality. Among the 21 patients with normal echocardiographic findings, increased QRS voltage was noted in 3, right bundle branch block in 1, old anteroseptal infarction in 1, coronary sinus rhythm in 1 and nonspecific ST-T wave abnormality in 1. Among the 28 cocaine abusers with echocardiographic left ventricular hypertrophy, increased QRS voltage was present in 6, nonspecific ST-T wave abnormalities in 4, intraventricular conduction delay in 3 and abnormal R wave progression in 3. Among the 11 patients with a segmental wall motion abnormality, nonspecific ST-T wave abnormalities were present in 3 and old inferior wall infarction was present in 1 patient.

Discussion

Cocaine-associated myocardial ischemia, infarction, coronary spasm, pulmonary edema, arrhythmias and sudden death have been reported (1,3,4). However, few studies have prospectively evaluated cardiac manifestations of long-term cocaine abuse, and the prevalence of cardiac abnormalities in asymptomatic cocaine abusers is unknown. The results of this study demonstrate that long-term cocaine abuse is associated with significant left ventricular hypertrophy and segmental wall motion abnormalities, whereas global ven-

Table 3. Left Ventricular Filling Variables in Cocaine Abusers (Group 1), Normal Volunteers (Group 2) and Control Patients (Group 3)

	Group 1	Group 2	Group 3	F
Heart rate (beats/min)	66 ± 9	67 ± 9	62 ± 9	1.33
E (cm/s)	83 ± 17	82 ± 13	85 ± 14	0.13
A (cm/s)	55 ± 12	53 ± 8	55 ± 7	0.20
E/A ratio	1.59 ± 0.58	1.58 ± 0.40	1.56 ± 0.30	0.01
DHT (ms)	86 ± 12	86 ± 15	87 ± 13	2.83
PFR (SV/s)	4.21 ± 0.67	4.45 ± 0.86	4.31 ± 0.55	0.70

All differences between groups were nonsignificant. A = velocity of atrial filling; DHT = deceleration half-time; E = velocity of early filling; PFR = peak filling rate; SV = stroke volumes.

Table 4. Left Ventricular Diastolic Filling Variables in 52 Cocaine Abusers With and Without Left Ventricular Hypertrophy (LVH)

	LVH (n = 28)	No LVH (n = 24)
Heart rate (beats/min)	67 ± 11	64 ± 7.5
E (cm/s)	78 ± 15	87 ± 19
A (cm/s)	56 ± 11	53 ± 13
E/A ratio	1.45 ± 0.45	1.72 ± 0.68
DHT (ms)	85 ± 11	87 ± 11
PFR (SV/s)	4.24 ± 0.49	4.17 ± 0.82

All differences between groups were nonsignificant. A = velocity of atrial filling; DHT = deceleration half-time; E = velocity of early filling; PFR = peak filling rate; SV = stroke volume.

Table 5. Prevalence of Echocardiographic Abnormalities n(%) in Cocaine Abusers (Group 1) Compared With Normal Volunteers (Group 2) and Control Patients (Group 3)

	Group 1 (n = 52)	Group 2 (n = 14)	Group 3 (n = 14)
LV mass index >134 g/m^2	28 (54)	0	0
Septum >1.1 cm	26 (52)	0	1 (7)
Posterior wall >1.1 cm	26 (52)	0	1 (7)
LV ejection fraction <0.45	2 (4)	0	0
LV dimension >6.0 cm	5 (10)	0	1 (7)
LV dimension >3.1 cm/m^2	5 (10)	0	0
E/A ratio <1	2 (4)	0	0
Segmental wall motion abnormality	11 (21)	0	0
Mitral valve prolapse	2 (4)	0	0

Data are presented as number (percent) of patients. Abbreviations as in Tables 2 and 3.

tricular systolic function and diastolic filling are normal in most patients.

Electrocardiographic abnormalities. The prevalence of abnormal ECG findings was high. To our knowledge, there are no other reports describing ECG findings in asymptomatic cocaine abusers. Even when sinus bradycardia, increased QRS voltage and early repolarization were classified as normal variants, 29% of patients had abnormal ECG findings. ST segment elevation due to early repolarization was present in 50%, an observation that may in part be explained by the large proportion (79%) of young black men in the study group. However, previous studies (17) have reported only a 27% prevalence of early repolarization in healthy, young black men. Five other patients in our study had ST segment elevation that was not typical of early repolarization, and ischemia could not be excluded. Our findings are similar to those of Gitter et al. (18), who reported the ECG findings in 100 cocaine abusers who presented to the emergency room with chest pain but were found not to have myocardial infarction. ST segment elevation was present in 43 patients and was attributed to early repolarization in 25, left ventricular hypertrophy in 7, acute myocardial injury in 8, pericarditis in 2 and old infarction in 1 patient. An explanation for the higher prevalence of ST segment elevation in these patients may be reversible silent ischemia. Nademanee et al. (19) reported that cocaine abusers frequently develop silent myocardial ischemia, manifested as episodes of ST segment elevation during ambulatory ECG monitoring performed during the 1st week of withdrawal from cocaine.

Right bundle branch block, first-degree AV block, left axis deviation and ectopic atrial rhythm were seen in one subject (2%) each. These abnormalities are present in <1% of healthy normal young men (20,21). Larger numbers of subjects are needed to determine whether the higher prevalence is significant. The prevalence of old myocardial infarction and nonspecific ST-T wave changes in this study was similar to that reported by Gitter et al. (18). Electrocardiographic abnormalities are common in asymptomatic cocaine abusers.

Left ventricular hypertrophy and diastolic filling. Left ventricular hypertrophy is usually defined as left ventricular mass >134 g/m^2. By this criterion, 54% of the cocaine abusers had left ventricular hypertrophy. Previous studies suggest that cocaine abuse may increase left ventricular mass. In an autopsy study, Virmani et al. (5) reported that 8 of 40 patients who died of cocaine-associated causes had increased heart weight. Left ventricular hypertrophy diagnosed by electrocardiography was reported (18) in 16% of cocaine abusers who were admitted to the hospital because of chest pain. Brickner et al. (22) studied 30 cocaine abusers with echocardiography; 13 (43%) had increased posterior wall thickness (≥1.2 cm). Left ventricular mass was increased when compared with that in normal control subjects, but the usual criterion for the diagnosis of left ventricular hypertrophy (mass index >134 g/m^2) was not met. The reason for higher left ventricular mass measurements in our study is unclear but it probably reflects differences in the study group and the timing of the study. It is possible that more potent forms of cocaine may lead to more hypertrophy; all the subjects in this study used crack cocaine. Numerous studies (23–25) have shown that removal of the inciting cause (discontinuing exercise, weight reduction, control of hypertension) will at least partially reverse hypertrophy. Thus, the interval between the echocardiogram and the last dose of cocaine may also have a role. In this study 75% of cocaine abusers had cocaine metabolites detected in the urine, indicating recent use.

The demonstration of left ventricular hypertrophy in cocaine abusers does not prove a cause and effect relation. However, the pharmacologic effects of cocaine are likely to produce left ventricular hypertrophy. Cocaine blocks the presynaptic reuptake of the neurotransmitters norepinephrine and dopamine, producing an excess of transmitter at the postsynaptic receptor sites (1). Activation of the sympathetic nervous system by this mechanism produces vasoconstriction, an abrupt increase in arterial blood pressure and tachycardia. Gottdiener et al. (26) recently reported that 64% of normotensive men with a hypertensive response during exercise have left ventricular hypertrophy. Thus, intermittent elevation of blood pressure may lead to left ventricular hypertrophy. Long-term administration of norepinephrine has been shown to produce left ventricular hypertrophy even

Table 6. Selected Measurements in Cocaine Abusers Classified According to Concomitant Alcohol Use

	Heavy (n = 18)	None/Rare (n = 19)	Intermediate (n = 14)
LV mass	264 ± 63	269 ± 55	281 ± 88
LV mass index	137 ± 35	144 ± 28	143 ± 38
LV dimension	5.3 ± 0.63	5.1 ± 0.55	5.1 ± 0.55
LV EF	66 ± 11	62 ± 12	64 ± 11

Data are expressed as mean value ± SD. All differences among the three groups were nonsignificant. EF = ejection fraction; LV = left ventricular.

in the absence of blood pressure elevation (27). Further studies are needed to elucidate the mechanism of left ventricular hypertrophy in cocaine abuse. Differential diagnosis of unexplained left ventricular hypertrophy in young men should include cocaine abuse.

Left ventricular diastolic filling abnormalities are common in patients with left ventricular hypertrophy due to hypertension, obesity and aortic stenosis (28,29). However, not all forms of hypertrophy lead to an abnormal filling pattern. Highly trained athletes with physiologic hypertrophy have normal diastolic function (30,31). Left ventricular diastolic filling in cocaine abusers has not been previously studied. This study demonstrates that the Doppler-derived diastolic filling pattern is normal in long-term cocaine abusers, even in the presence of left ventricular hypertrophy.

Left ventricular wall motion abnormalities. Myocardial infarction is reportedly the most common abnormality associated with cocaine abuse (4–6). However, this prevalence may be due to the fact that most reports are of isolated cases or series of cases in which patients sought medical attention after a cardiac event. Nademanee et al. (19) reported that episodes of transient myocardial ischemia manifesting as ST segment elevation are common in cocaine abusers during the 1st week of withdrawal. In our study 11 (21%) of cocaine abusers had segmental wall motion abnormalities and none reported chest discomfort. A cine loop format was used to evaluate left ventricular wall motion, and this technique may have increased the sensitivity for the detection of abnormalities. One subject had global hypokinesia of the left ventricle. Isolated cases of dilated cardiomyopathy have been reported (32) to complicate cocaine abuse. In our study a dilated left ventricle was noted in five (10%) of cocaine abusers and a depressed left ventricular ejection fraction in two (4%). In the only previous report (22) we found describing echocardiographic findings in asymptomatic cocaine abusers, increased left ventricular mass was found in the 30 cocaine abusers studied, but no segmental wall motion abnormalities were reported. Studies of a larger number of patients are needed to provide a better estimate of the prevalence of wall motion abnormalities in asymptomatic cocaine abusers.

Role of concomitant alcohol abuse. In the Framingham Study, alcohol use was demonstrated to be independently associated with left ventricular mass (33). Because many cocaine abusers also used various amounts of alcohol, it is important to demonstrate that the cardiac abnormalities are not secondary to alcohol use. The control group consisting of normal volunteers (physicians and nurses) did not smoke and consumed only a small amount of alcohol. Accordingly, another control group consisting of patients from the psychiatric service were also studied; these control patients had a pattern of smoking and alcohol use similar to that of the cocaine abusers. Cocaine abusers had a higher left ventricular mass than that of subjects in both control groups. Among the cocaine abusers, the amount of alcohol consumed did not affect the degree of left ventricular hypertrophy. Estimation of alcohol consumption by history is not a reliable method. However, among the cocaine abusers there was no significant difference in left ventricular mass between those who used very little or no alcohol and those who drank moderately.

Limitations of the study. A relatively small number of cocaine abusers were studied. "Street cocaine" may contain various amounts of active cocaine and contaminants that may lead to cardiac complications. Although our intention was to recruit subjects who abused all forms of cocaine, all enrolled subjects were long-term crack cocaine abusers. The potency of the cocaine and the duration of abuse may be significant, and our findings may not be applicable to all forms of cocaine abuse. The control subjects were not matched by race, and the proportion of black subjects in the control groups was smaller. However, as in other races, young, asymptomatic and normotensive men of the black race also have a very low likelihood of heart disease (34). The left ventricular mass of black cocaine addicts was not higher than that of white and Hispanic addicts. However, black hypertensive patients are more prone to develop left ventricular hypertrophy (35), and it is possible that there are racial differences in the cardiac response to cocaine.

This study evaluated the cardiac abnormalities at one point in time; most of the cocaine abusers had documented recent use of cocaine. Abnormalities like left ventricular hypertrophy and coronary artery spasm may resolve with abstinence from cocaine. Diastole is a complex phenomenon and the mitral flow velocity curves are influenced by many interrelated factors. A normal Doppler-derived diastolic filling pattern does not assure normal diastolic filling.

Conclusions. Electrocardiographic and echocardiographic abnormalities are common in chronic crack cocaine abusers. They were characterized in our patients by frequent occurrence of left ventricular hypertrophy with a normal Doppler-derived diastolic filling pattern and occasional occurrence of segmental wall motion abnormalities; the left ventricular ejection fraction was normal in most subjects. The concomitant use of alcohol did not alter the prevalence of these abnormalities.

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